A Case of *Staphylococcus aureus* Enterocolitis: A Rare Entity

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Staphylococcus aureus is a common organism found in nosocomial and postoperative infections. Enterocolitis caused by this organism can be severe and sometimes fatal.[1] In the 1950s and 1960s, *S. aureus* was implicated as a major pathogen in postoperative and antibiotic-associated enterocolitis.[2-5] Although it continued to be a problem in Japan,[1,6-8] its incidence in the United States has diminished.[3] The clinical significance of *S. aureus* isolated from stool is unknown, as it is often seen unrelated to the clinical picture. We report a case of a patient with hemorrhagic enterocolitis who was initially suspected of having inflammatory bowel disease, but, based upon the diagnostic evaluation, was found to have *S. aureus* enterocolitis.

Case Report

A 49-year-old male hospital plumber with a history of hemorrhoids presented to an outside hospital with abdominal cramps and diarrhea. He was diagnosed with a gastrointestinal infection and was started on levofloxacin, metronidazole, and proton pump inhibitor therapy. The patient was discharged 2 days later but returned within 24 hours with worsened symptoms and bloody diarrhea. Computed tomography (CT) scan revealed thickening of the duodenum and jejunum (Figure 1), and enteroscopy revealed an ulcerated duodenum and jejunum with necrosis (Figure 2). Pathology showed moderate-to-severe acute and chronic inflammation and acute inflammatory exudate.

The patient was transferred to Allegheny General Hospital in Pittsburgh, Pennsylvania. On admission, he was in mild distress and tachycardic and continued to complain of his initial symptoms. His abdomen was soft and mildly distended, with slightly hypoactive bowel sounds and mild diffuse tenderness. Relevant laboratory values included C-reactive protein of 13.9 mg/dL, erythrocyte sedimentation rate of 45 mm/hr, white blood cells of 24,100/mcL, neutrophils of 54,000/mcL, and bands of 40,000/mcL. Stool microscopic examination and culture were positive for many polymorphonuclear neutrophils and *S. aureus*. Stool culture was negative for *Camplobacter*, *Shigella*, *Salmonella*, and *Yersinia*. Repeat CT scan showed thickened terminal ileum and right colon, and work-up for hypercoagulable state and ischemia were negative. The patient was started on intravenous fluids and continued on flouroquinolone, metronidazole, and proton pump inhibitor therapy. Surgical consultation was obtained. The patient's symptoms gradually improved, but bandemia persisted. Upper gastrointestinal small bowel follow-through showed flocculation of the ileum. On Day 10, a repeat enteroscopy was normal to the jejunum (Figure 3A). Also on Day 10, a flexible sigmoidoscopy showed erythema, congestion, ulceration, friability, and exudate from the rectum to the splenic flexure (Figure 3B–D). On

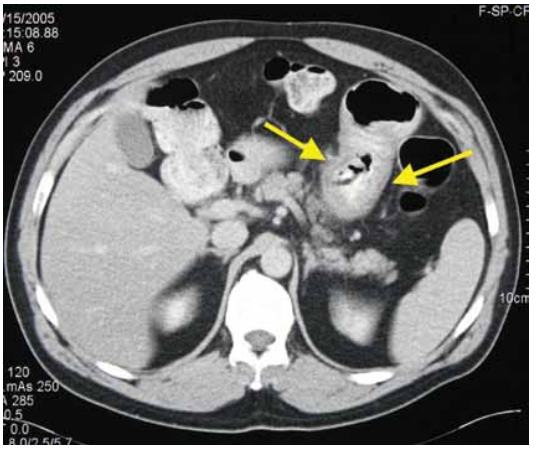

Figure 1. Abdominal computed tomography scan on Day 1 showed jejunal distention and thickening and no focal mass lesions or obstruction.

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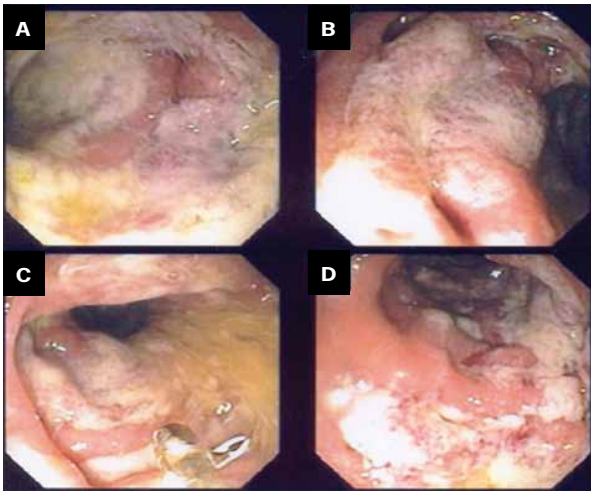

Figure 2. Enteroscopy on Day 1 showed the duodenum with erythema, congestion, ulceration, friability, and inflammatory exudate (**A** and **B**) and the jejunum with erythema, congestion, ulceration, friability, necrosis, and inflammatory exudate (**C** and **D**).

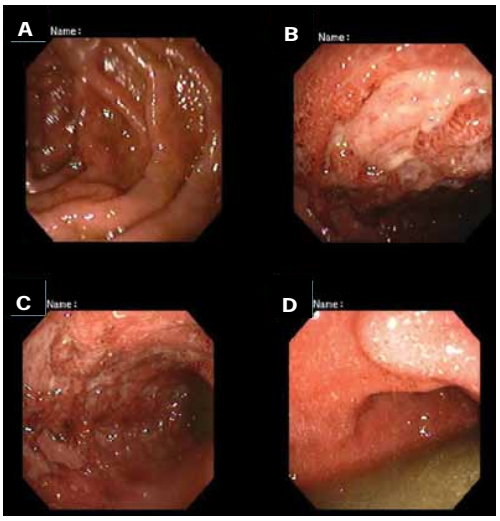

Figure 3. Enteroscopy and sigmoidoscopy on Day 10 showed diffuse continuous erythema, congestion of the mucosa, and friability in the proximal jejunum (**A**); erythema, congestion, ulceration, friability, and exudate in the descending colon (**B** and **C**); and erythema, congestion, ulceration, friability, and exudate in the rectum (**D**).

Day 12, the patient's clinical status worsened, and he was taken to surgery. Exploratory laparotomy revealed a toxic megacolon, and a gram stain from the surgical specimens revealed gram-positive cocci in clusters (Figure 4). Histopathology from the colon and ileum resections showed chronic active colitis with cryptitis and crypt abscesses, focal mucosal inflammation and transmural acute inflammation, and ulceration and necrosis (Figure 5).

Postoperatively, the patient's symptoms resolved. He was discharged without antibiotic therapy and remained in clinical and endoscopic remission at the 6- and 12-month follow-up visits.

Discussion

S. aureus necrotizing enterocolitis has most commonly been described in patients with severe underlying disease, previous gastrectomy, use of antipeptic ulcer drugs, recent antibiotic use, and in patients previously colonized with methicillin-resistant *S. aureus*.[3,4,9,10] Symptoms of *S. aureus* necrotizing enterocolitis include nausea, vomiting, diarrhea associated with fever, and evidence in the stool of an acute inflammatory process affecting the gut mucosa (ie, pus, mucus, or blood).[5,9] Additionally, dysentery with crampy abdominal pain and tenesmus may be present. Radiologic findings are nonspecific but may show bowel thickening. Endoscopic findings are also nonspecific and may include patchy erythematous areas, pseudomembranous disease, and ulcerations with necrosis.[5,9] Pathologic findings include acute patchy necrotizing disease, which can progress rapidly to complete structural necrosis and

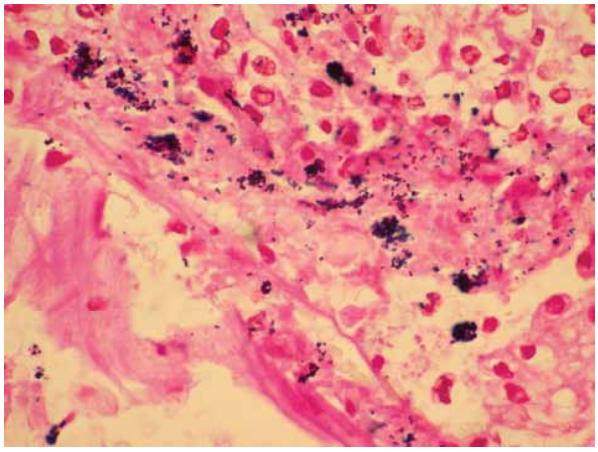

Figure 4. Gram stain revealing gram-positive cocci in clusters.

segmental gangrene. The diagnosis of *S. aureus* enterocolitis is often made by clinical history and exclusion of other enterocolitides; gram stain of biopsy specimens; and isolation of *S. aureus* from the stool.

In this case report, the patient had received broad-spectrum antibiotics and empiric acid suppression and was at a high occupational risk for a nosocomial infection. Although endoscopic and pathologic findings may suggest Crohn's disease, necrosis on pathology and endoscopic remission of duodenal and jejunal pathol-

ogy within days of antibiotic therapy are not typical findings of acute and chronic Crohn's disease. Signs and symptoms consistent with *S. aureus* necrotizing enterocolitis in this patient included bloody diarrhea, thickening of the duodenum and jejunum on CT, and an ulcerated duodenum and jejunum and necrosis on enteroscopy. Furthermore, endoscopic and surgical specimens revealed gram-positive cocci in clusters, and pathologic findings showed ulceration and necrosis. Medical therapy with oral vancomycin has been shown to be effective,[3,4,6] but surgery may be required for more advanced disease, such as in the present case. This case helps bring awareness to the existence of a rare entity.

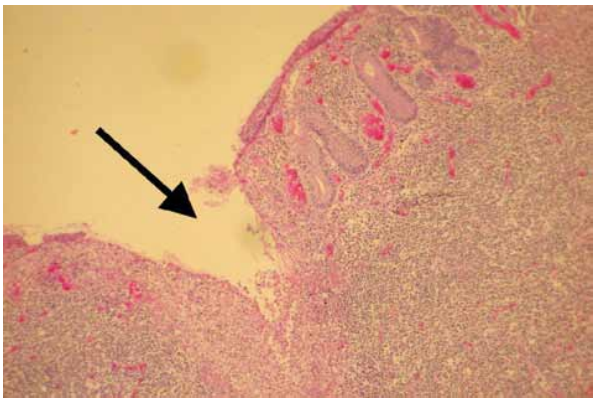

Figure 5. Colon with ulceration and necrosis.

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Review

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First recognized almost 70 years ago,[1] enterocolitis due to *Staphylococcus aureus* has been described as both a complication of antibiotic therapy and as occurring in individuals with predisposing conditions but no previous antibiotic treatment. Sporadic cases and outbreaks have been reported in infants since the 1940s, with prematurity and low birth weight as the major predisposing factors. In adults and noninfant children, staphylococcal enterocolitis is usually associated with prior use of antimicrobials (particularly fluoroquinolones), recent abdominal surgery, prior proton pump inhibitor therapy, and immune-compromising conditions such as advanced age, immunosuppressive therapy, and HIV infection.

However, following the identification of *Clostridium difficile* and its toxins as the primary cause of pseudomembranous colitis in the 1970s, the role of *S. aureus* in antibiotic-associated colitis has been downplayed. As a result, awareness of staphylococcal enterocolitis has diminished in the medical community, to the extent that *S. aureus* is not universally considered to be a potential etiology of nosocomial diarrhea.[2] Consequently, stool culture may not be requested in cases of healthcare-associated diarrhea, even after *C. difficile* has been excluded. However, as demonstrated by the case reported by Thakkar and Agrawal,[3] staphylococcal enterocolitis has not disappeared; thus,

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